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LETTERS TO THE EDITOR

Closure of Incompletely Surgically-Ligated Left Atrial Appendage in Reducing Stroke Risk

We read with great interest the *Image in Intervention* by Matsumoto et al. (1), published in the February 2013 issue of *JACC: Cardiovascular Interventions*. In it, the authors describe a procedure for percutaneous endocardial occlusion of incompletely surgically ligated left atrial appendage (ISLL) using a septal occluder device guided by transesophageal echocardiography (TEE) and angiography in a patient with atrial fibrillation (AF) previously treated with a surgical maze procedure, mitral and tricuspid valve repair, and attempted surgical ligation of the left atrial appendage. In addition, they argue that the proposed approach can "reduce the risk of thromboembolism and stroke."

Similarly, our group has recently published a paper on the safety and feasibility of this technique (2). Although the clinical significance of ISLL warrants further investigation, it is believed to be associated with an increased risk of thromboembolism. Moreover, it is believed that ISLL may actually be worse than no occlusion at all, given that reduced blood flow in and out of a "stenotic" left atrial appendage may in fact promote a higher risk of thrombus formation inside this structure (3). Consistent with this, 3 patients in our series presented with an early embolic event following ISLL despite a $CHADS_2$ score ≤ 1 and antiplatelet therapy. In addition, we have observed an inverse relationship between embolic stroke risk and the size of the ISLL neck diameter in our patients.

We, too, have not observed any embolic events in our small cohort of AF patients who underwent percutaneous endocardial ISLL occlusion, subsequently off oral anticoagulation therapy, during 8 ± 2 months of follow-up. Nevertheless, we have remained cautious in offering hasty conclusions regarding long-term stroke risk reduction through such an approach. An important element that needs to be taken into consideration is the underlying disease substrate. That is, although atrial thrombi likely originate inside the left atrial appendage in nearly 90% of patients with nonvalvular AF, the same is true for <50% of patients with valvular AF, as shown in a recent systematic review of 34 studies (4). Hence, firm conclusions regarding stroke risk reduction in the setting of AF and cardiac valvular pathology derived purely on the basis of a single case report seems premature.

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REFERENCES

1. Matsumoto T, Nakamura M, Yeow WL, Siegel RJ, Kar S. Transcatheter left atrial appendage closure after incomplete surgical ligation. J Am Coll Cardiol Intv 2013;6:e11–2.
2. Aryana A, Saad EB, d'Avila A. Left atrial appendage occlusion and ligation devices: what is available, how to implement them, and how to manage and avoid complications. Curr Treat Options Cardiovasc Med 2012;14:503–19.
3. Fuller CJ, Reisman M. Stroke prevention in atrial fibrillation: atrial appendage closure. Curr Cardiol Rep 2011;13:159–66.
4. Mahajan R, Brooks AG, Sullivan T, et al. Importance of the underlying substrate in determining thrombus location in atrial fibrillation: implications for left atrial appendage closure. Heart 2012;98:1120–6.

Reply

We appreciate the comments of Drs. Aryana and d'Avila regarding our paper "Transcatheter Left Atrial Appendage Closure after Incomplete Surgical Ligation" (1) and their own experience in similar circumstances (2). Great minds do think alike.

We completely agree that in patients with valvular atrial fibrillation (AF), the substrate is different, with ~44% of thrombus in the left atrial appendage (LAA) (3). However, the quoted cohort composed almost entirely of patients with rheumatic mitral stenosis, much different from our patient, who underwent a successful surgical mitral valve repair for nonrheumatic mitral regurgitation. The patient was left with only minor residual mitral regurgitation after the repair.

We also agree with the authors that an incomplete surgical closure with a "stenotic" orifice could promote blood stagnation, but when coupled with reduced LAA flow velocities from AF, the thromboembolism risk maybe further accentuated. In the prospective, randomized clinical trial of the Watchman device (Atritech, Inc., Minneapolis, Minnesota), a residual gap >5 mm is considered an unsuccessful closure, requiring continuation of the patient's anticoagulation (warfarin) (4). Therefore, we believe that if the original intent is to perform LAA exclusion and the risk of thromboembolism is unacceptable, "the residual communication secondary to incomplete surgical ligation can be closed percutaneously to reduce the risk of thromboembolism and stroke."

We report this case because it demonstrates a relatively simple solution and a clinically feasible approach to treat patients who have a residual LAA to left atrial communication after surgical ligation of the LAA. Additional clinical studies including of a large numbers of patients will be necessary to better elucidate whether the treatment approach we have shown to be feasible will in fact decrease thromboembolic complications. We appreciate the authors and editor for giving us an opportunity to clarify our statement.

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